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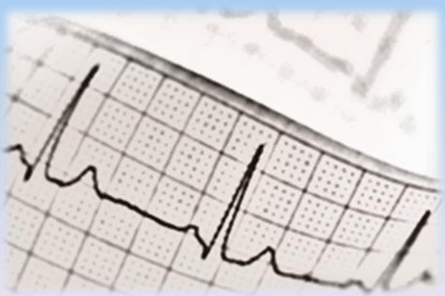
The Acute ECG- Interpreting changes beyond the ST segment

Nicholas Tayler, Andrew R. J. Mitchell



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Introduction

This guide is designed to guide you through the step by step process of **clinical** ECG interpretation. A good knowledge of cardiac physiology is assumed. It is intended to promote a broad approach to **myocardial infarction**, thinking about the ECG as a whole and not just the ST segment.

This is not designed to be a diagnostic device, but simply to teach the principles behind recognising different ECG changes in a systematic and clinical way: rhythm, P waves, QRS complex, ST segment, T waves and finally other morphology.

ECG interpretation will become ‘pattern recognition’ and this guide is designed to help show you the patterns to look for and how to interpret them.

About the authors:

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Dr Andrew Mitchell is a consultant cardiologist at Jersey General Hospital.

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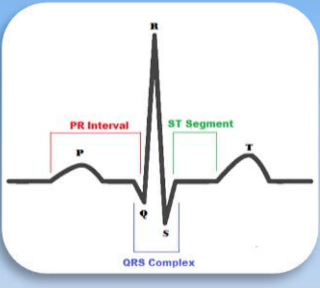
Normal values



P wave	0.08-0.10s	(2-2.5mm)
PR interval	0.12-0.20s	(3-5mm)
QRS	<0.12s	(<3mm)
$Q\text{-}T_c$	<0.45s	(<11mm)
QT_c	$QT/\sqrt{RR}$	

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Step 1- Demographics

- Is it the right patient?
- Is it the right ECG?
- When was the ECG taken?
- Do they have any old ECGs for comparison?
- Is the ECG of sufficient quality for interpretation? E.g. minimal artefact from movements.

Continue

Symptoms

- Always treat the patient.

- What symptoms do they have?
- E.g. Cardiac chest pain will have an impact on your diagnosis and interpretation.

- If the patient does not have a pulse then the key rhythms to recognise are:
- Ventricular Tachycardia
- Ventricular Fibrillation
- Pulseless Electrical Activity

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Is the ECG Regular?

- Are the spaces between QRS complexes the same each time?
- (How do I check this?)

YES: Regular ECG

No: Irregular ECG

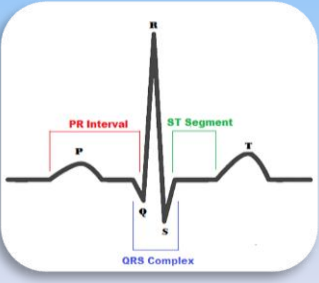
How to check if regular?

- Take a piece of paper and align it to the rhythm strip.
- Make a mark at each R wave for 5 complexes.
- Move this along the rhythm strip and see if the complexes line up to the marks on the paper.
- If all complexes match up this is regular, if a pattern of complexes line up it is regularly irregular.

BACK

Are there P waves?

- P waves represent the atria contracting in a coordinated fashion:



YES: P waves present

NO: P waves absent

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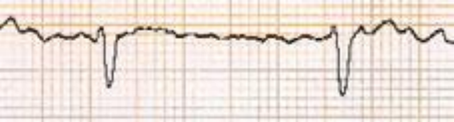
Atrial fibrillation

- The atria are contracting in a non-uniform manner like a ‘bag of worms’ reducing cardiac output by around 25%



- AF may be longstanding and asymptomatic and the patient may be on anticoagulants to reduce stroke risk.
- However it may be new onset and this might be a sign of **myocardial infarction**
- The rest of the ECG should be interpreted in a standard manner, as other morphology might be present.

NEW onset AF

Continue to examine ECG

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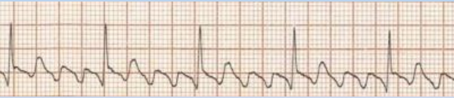
New onset Atrial Fibrillation

- Atrial fibrillation has many causes, including coronary heart disease, valvular heart disease, hypertension or an electrolyte imbalance.
- Therefore a thorough cardiac examination should be performed.
- Consider blood tests (urea and electrolytes) to exclude electrolyte disturbance. Also consider measuring troponin levels if associated with chest pain.
- The rest of the ECG should be analysed to determine any ischaemic or electrolyte induced changes.

Continue to examine ECG

Saw tooth P waves?

- Are P waves ‘saw tooth’ in pattern? (best seen in V_1 and II)
- Is the rate rapid (around 150bpm)?
- These are sometimes known as F waves.



YES: Saw tooth P waves

NO: Normal P waves

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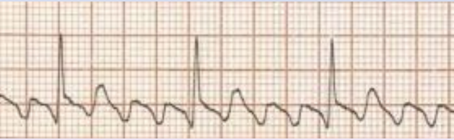
Atrial flutter

- Ventricular contraction may be regular or irregular.
- Atria are rapidly contracting at around 200-300 bpm.
- Not all these ‘P waves’ (also known as F waves) can be conducted through the AV node
- Therefore ventricular rate is lower. One example (below) is a 3:1 conduction where ventricles contract after every 3 P waves meaning the rate is c.100bpm.



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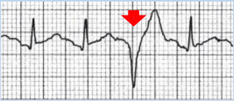
Do P waves relate to QRS complexes?

- Are the majority of P waves followed by a QRS complex?
- However are occasional P waves not followed by a QRS?
- Are ventricular ectopics present: broad complex, premature QRS complexes without an obvious preceding P wave?



YES: Most P → QRS

NO: P unrelated to QRS

Ventricular Ectopics

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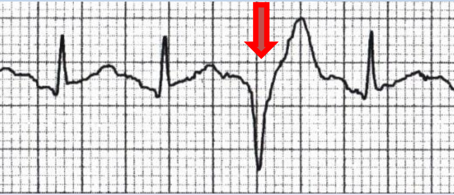
Ventricular Ectopics

- These are a normal electrical phenomenon and do not usually require investigation
- They can make an ECG appear misleading, but once identified then they should be noted and the rest of the ECG should be examined as normal.
- The ST segment should be disregarded for the ventricular ectopic wave



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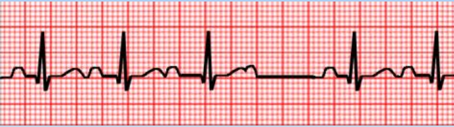
2nd Degree Heart Block

- This is a form of conduction block between the atria and the ventricles. It may lead to complete heart block and may be caused by **Myocardial Infarction**

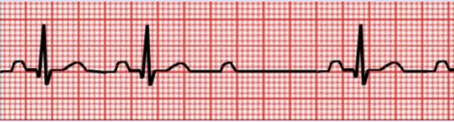
- Mobitz I block:

The PR interval becomes progressively longer, until a QRS complex is “dropped”



- Mobitz II block:

Not all P waves result in a QRS complex, some fail to stimulate the ventricles sometimes in a set pattern. (e.g. 3:1 block)



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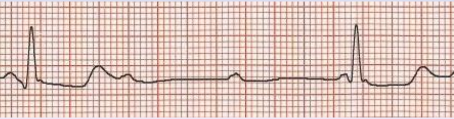
3rd Degree heart block

- Total or complete heart block
- May be caused by **myocardial infarction.**
- A failure of the conduction system to pass impulses from the atria to the ventricles.
- Therefore ventricles contract spontaneously in a ‘ventricular escape rhythm’ of around 40bpm
- The QRS complex is broad when it is of ventricular origin
- High risk patients may need admission and consideration of temporary pacing



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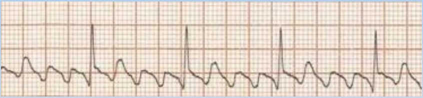
Regular ECG: Saw tooth P waves?

- Are P waves 'saw tooth' in pattern? (best seen in V_1 and II)
- Is the rate rapid (c. 150bpm)?

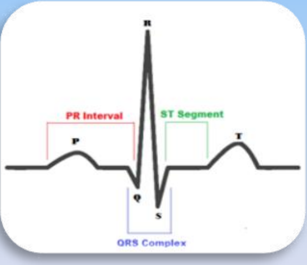


YES: Saw tooth P waves

NO: Normal P waves or P waves not visible

PR Interval

- Is the PR Interval less than 200ms? (5 small squares)
- If P waves not visible follows PR<200ms



YES: PR < 200ms

NO: PR > 200ms

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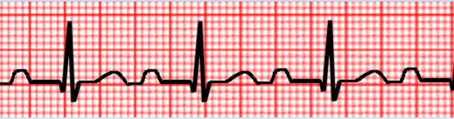
1st Degree Heart Block

- PR interval greater than 200ms
- May rarely be caused by **inferior MI** (if symptomatic), but also by athletic training, increased vagal tone, hypokalaemia and many others.
- Does not require specific treatment.
- NB 5% of medical students will have 1st degree heart block!



Continue to assess ECG

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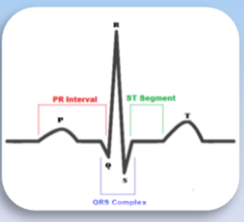
Narrow QRS Complex?

- Is the QRS complex < 120ms? (3 small squares)
- A narrow QRS complex represents normal, rapid conduction along the his-purkinje system.



YES: QRS < 120ms

NO: QRS > 120ms

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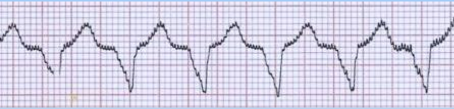
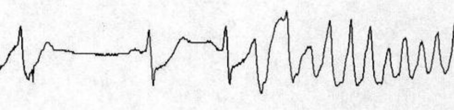
Broad complex: Is it an emergency rhythm?

Ventricular Tachycardia:



Ventricular Fibrillation:



No: Not VF or VT

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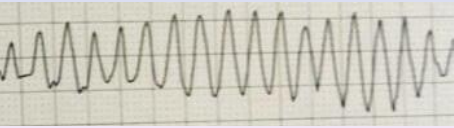
Ventricular Fibrillation

- Ventricular fibrillation is uncoordinated, random contraction of the ventricles at an uncontrolled rate
- Cardiac output is therefore lost and the patient will not have a pulse.
- It is one of the shockable rhythms and so it is essential to recognise in order to defibrillate.
- Another shockable rhythm is pulseless ventricular tachycardia
- It may be as a result of **myocardial infarction**



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Pulseless Electrical Activity

- If the patient does not have a pulse, but has electrical activity then this is known as pulseless electrical activity
- It directs you to the non-shockable rhythm algorithm for resuscitation
- It may resemble any rhythm (including sinus rhythm) but is not compatible with life: **treat the patient and not the ECG**

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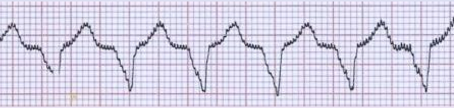
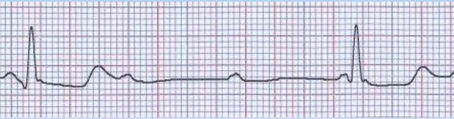
Ventricular Tachycardia

- This is a cardiac emergency
- Ventricular Tachycardia can degenerate into ventricular fibrillation and sudden cardiac death.
- Equally ventricular tachycardia may not produce a pulse and therefore requires urgent ABC assessment and defibrillation.
- An automated emergency defibrillator (AED) may not automatically shock this rhythm as the machine does not know if the patient has a pulse- therefore recognition is essential in order to resuscitate



Complete heart block?

- Are P waves present that do not relate to the broad QRS complexes?
- Is the rate slow (around 40bpm)?



YES: P unrelated to QRS

NO: P → QRS or P not visible

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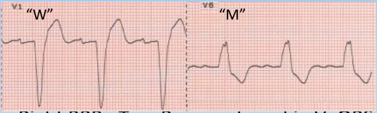
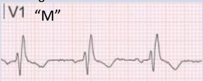
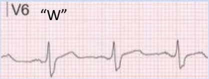
Bundle Branch Block

- P waves may be ‘hidden’ behind QRS
- This is a broad complex rhythm caused by a delay in conduction along the his-purkinje fibres, either in the left or right bundle.
- Left BBB= dominant S wave in V_1 and broad R waves in V_6
- WiLLiaM: W shape V_1 and M shape in V_6



- Right BBB= Two R waves found in V_1 QRS and wide S wave in V_6
- MaRRoW: M shape in V_1 and W shape in V_6





Left Bundle Branch Block

Right Bundle Branch Block

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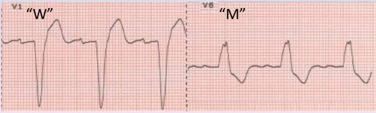
Left Bundle Branch Block

- Caused by block of the LEFT bundle, therefore the left ventricle is delayed in contraction, as the impulse must travel from the right ventricle
- New LBBB is often caused by organic heart disease (rarely idiopathic) which prompts careful investigation.
- Causes include **myocardial infarction**, ischaemia, cardiomyopathy and aortic stenosis.
- Due to the morphology of QRS complexes, the ST segment can appear raised. However this is difficult to judge and Sgarbossa Criteria should be used to evaluate whether a **myocardial infarction** has occurred



http://www.heartforlife.co.uk/Heart-Conditions/Left-Bundle-Branch-Block/

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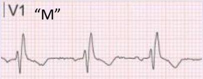
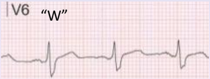
Right Bundle Branch Block

- Contraction of the right ventricle is delayed due to a block in the right bundle
- Therefore the right ventricle is depolarized by impulses travelling from the left ventricle and so contracts **after** the left ventricle.
- This produces a second R wave, forming an M shape in leads V_1-V_3
- RBBB can be found in healthy individuals, but may be a sign of underlying pathology, including **myocardial infarction.**
- Therefore symptoms, onset and tests such as troponin may be useful.
- ST Segments may appear raised in V_1-V_3 so these leads should not be used for routine evaluation.
- However ST segments in limb leads can be used, in conjunction with symptoms and troponin, to assess for possible **MI**





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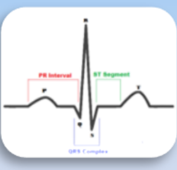
Is the ST Segment Elevated/ Depressed?

- Is the area between the S wave and T wave raised, depressed or normal (isoelectric)?
- ST elevation may be accompanied by reciprocal ST depression in other leads.



ST Elevation

ST Depression

ST Normal

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Acute Myocardial Infarction

- ST elevated myocardial infarction (STEMI) is the classical presentation of myocardial infarction.
- It represents full thickness ischaemia of the myocardium due to complete block of the relevant coronary artery- anterior, lateral or posterior territory MI.
- It is often accompanied by ST depression in reciprocal areas of partially ischaemic myocardium
- Management includes percutaneous intervention (PCI) to remove blockages from arteries

Pathophysiology

Territory

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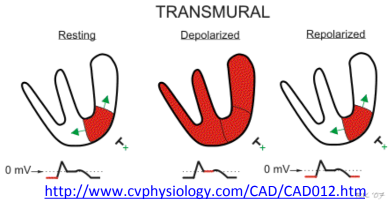
Pathophysiology

- The pathophysiology of the ST elevation is a result of hypoxia, which reduces intracellular ATP formation due to reduced aerobic respiration. This reduces the effect of the Na^+/K^+ ATPase pump, reducing K^+ being pumped into the cell.
- This causes the infarcted region to become depolarized continuously due to lack of ion movements.
- This produces depolarization currents that are travelling AWAY from the electrode, artificially lowering the baseline
- Therefore when an impulse occurs the ST segment *appears elevated*



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STEMI- Territory

- The area of infarction depends on which coronary artery has become occluded:
- The left anterior descending artery (LAD) occlusion causes an anterior STEMI which has the worst prognosis
- Anterior STEMI can be recognised by ST elevation in leads V_1-V_6 with reciprocal ST depression in III and aVF

- Inferior STEMI (40-50% MI) have lower mortality (2-9%) and are mostly due to Right coronary artery occlusion.
- ST elevation can be seen in the inferior leads II, III and aVF

- Lateral STEMI is often part of LAD occlusion termed anterolateral MI. However small artery branch occlusion can cause an isolated lateral MI
- ST elevation is seen in I, aVL and V_5-V_6

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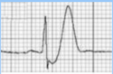
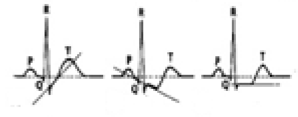
ST Depression

- ST depression is a sign of partial thickness ischaemia of the myocardium
- It may occur in isolation or as a reciprocal change in STEMI. If ST depression is present a careful check for ST elevation should be made.
- ST depression with a De Winter's T wave is a sign of STEMI:



- ST depression may be up sloping, which is not specific for cardiac ischaemia. Flat or down sloping ST depression is a sign of ischaemia:



Pathophysiology

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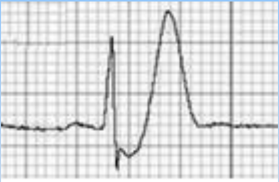
De Winter's T wave

- This is a sign of **anterior STEMI** *without* ST elevation.

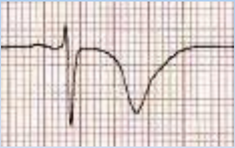


- It is characterised by ST depression and peaked T waves in leads V_1 to V_6
- It is seen in around 2% of left anterior artery occlusions and is under-recognised by physicians

STEMI

ST depression: T wave inversion?

- Are there inverted T waves present?
- Inverted T waves can be a normal variant in leads III, aVR and V_1



T wave inversion present

T wave inversion absent

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NSTEMI?

- Non ST elevation MI (NSTEMI) is a partial thickness infarction of the myocardium.
- Diagnosis is made by new (dynamic) ECG changes **combined with raised cardiac biomarkers in the blood (e.g. Troponin).**
- >2mm ST depression in ≥3 leads strongly indicates a non ST elevation myocardial infarction (NSTEMI)
- T wave inversion is a sign of cardiac ischaemia and so may support the diagnosis
- Dynamic changes without raised troponin levels indicate **unstable angina (UA).**
- NSTEMI, STEMI and UA together form Acute Coronary Syndrome (ACS)

NSTEMI

Unstable Angina

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NSTEMI

- Non-ST Elevated myocardial infarction is raised cardiac biomarkers with dynamic ECG changes and/or cardiac chest pain.
- Troponin T or I are components of cardiac muscle stored in myocytes. Cell death releases these into the blood stream pathologically and so can be detected by blood tests.
- T wave inversion is where repolarization occurs in the opposite direction to normal due to damage to the cardiac endothelium (the area of myocardium most likely to suffer ischaemic damage due to being the greatest distance from blood supply).
- If present with or without ST depression the suspicion of an NSTEMI is raised and Troponin levels should be measured.
- Management includes anti thrombotic treatment and early revascularisation.

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Unstable angina

- ECG changes without a raised blood biomarker are classified as unstable angina.
- Symptoms are typically:
  - Angina at rest
  - New onset severe angina
  - Crescendo of worsening angina over a short time period.

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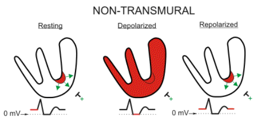
Pathophysiology of ST depression

- NSTEMI is partial thickness infarction of the myocardium
- The infarcted area is damaged and so leads to release of cardiac biomarkers into the blood that can be detected by a blood tests
- Partial thickness cell death results in an area of the myocardium becoming continuously depolarised as ATP is no longer available to pump Na^+/K^+ ions to polarise the membrane
- This sends waves of depolarization through surrounding tissue when the muscle is at rest. If this travels towards the electrode then this is detected as a positive deflection.
- Therefore the baseline is artificially raised, which means the ST segment *appears* lower than the baseline causing ST depression.



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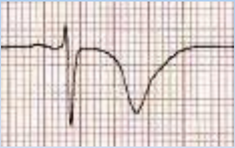
T wave inversion?

- Are there inverted T waves present?
- Inverted T waves can be a normal variant in leads III, aVR and V_1



[T wave inversion present]

[T wave inversion absent]

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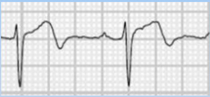
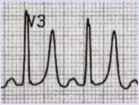
Other morphology?

- Biphasic T waves?
  - These are present in Wellen’s syndrome; a group of patients at high risk of anterior MI in the next 2-3 weeks



- Tall, tented T waves?
  - May be a sign of ischaemia or hyperkalaemia; risk of arrhythmia and cardiac death



Normal morphology

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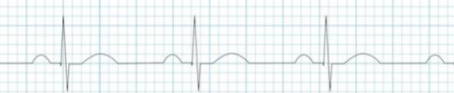
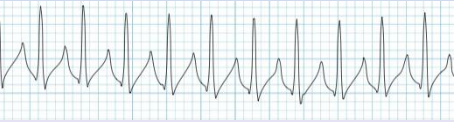
Sinus Rhythm

- Rate < 60 = sinus bradycardia
- Rate >100= sinus tachycardia



- Remember that P waves can appear hidden behind other complexes, such as in supraventricular tachycardia:



- IF IN DOUBT: Treat the patient and ask for senior help early!

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Acknowledgements

- Many thanks to Dr Andrew Mitchell for verifying the content of this resource, providing ECG examples and guidance throughout development.